BREAK POINT

2014 - ISSUE 10

FROM THE NEWSLETTER EDITOR'S DESK

Welcome to the October edition of *Breakpoint*. Following "In the news", in this issue, Joshua Ramsay from Curtin University, and Matthew O'Sullivan, Westmead Hospital, provide us with highlights of this year's International Symposium on Staphylococci and Staphylococcal Infections (ISSSI) conference in Chicago, a treat for those with an interest (as well as for others) in all things "Staphylococcal". Notices and a conference calendar follow. As always, feedback is warmly welcome.

Sharon Chen

ASA Breakpoint Editor,

On behalf of the ASA committee

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IN THE NEWS

Shortened TB treatment with moxifloxacin-containing regimen is not effective

Because of their bactericidal activity against *Mycobacterium tuberculosis*, the use of fluoroquinolones in an anti-tuberculous regimen was hypothesized to allow a shorter duration of treatment. However, shortening treatment with a moxifloxacin-containing regimen to four months was not effective in a phase III trial that included 1931 patients with uncomplicated, smear-positive pulmonary tuberculosis. Each of two moxifloxacin-containing regimens (isoniazid, rifampin, pyrazinamide, and moxifloxacin; or rifampin, pyrazinamide, ethambutol, and moxifloxacin) given for 17 weeks resulted in greater treatment failure and relapse compared with the standard control regimen of two months of isoniazid, rifampin, pyrazinamide, and ethambutol followed by four months of isoniazid and rifampin. This was despite shorter times to negative sputum cultures with the moxifloxacin regimens

Reference: Gillespie SH, Crook AM, McHugh TD, *et al*. Four-Month Moxifloxacin-Based Regimens for Drug-Sensitive Tuberculosis. N Engl J Med 2014.

IN THE NEWS (CONT'D)

Antimicrobial stewardship - are prophylactic antibiotics required before laparoscopic cholecystectomy
Multiple meta-analyses of randomized trials comparing antibiotic prophylaxis with no antibiotic or placebo prior to elective laparoscopic cholecystectomy have found no differences in the incidence of surgical site infection. These trials have generally included 50 to 175 patients in each arm and have used a single dose of antibiotics administered just prior to surgery. A subsequent large trial randomly assigned approximately 1000 low-risk patients undergoing elective laparoscopic cholecystectomy to no antibiotics or intravenous antibiotics given preoperatively and, 12 and 24 hours post operatively. Herein the incidence of surgical site infection was lower for those who received perioperative antibiotics (0.8 vs 3.7 percent without antibiotics). Despite the added cost of antibiotic administration, overall hospital costs were lower in the prophylaxis group. Further investigation is needed to determine whether the infectious risk reduction of additional antibiotic doses outweighs the risks of toxicity or bacterial drug resistance associated with increased antibiotic usage.

Reference: Matsui Y, Satoi S, Kaibori M, *et al*. Antibiotic prophylaxis in laparoscopic cholecystectomy: a randomized controlled trial. PLoS One 2014; 9:e106702.

ANTIMICROBIALS 2015 MEETING REGISTRATION WEBSITE OPEN

26 - 28 February 2015 , Brisbane Exhibition and Convention Centre. **www.antimicrobials2015.com**

On behalf of the Australian Society for Antimicrobials I would like to invite you to the Society's 16th Annual Scientific Meeting "Antimicrobials 2015" to be held at the Brisbane Exhibition and Convention Centre, Brisbane, on Thursday 26th - Saturday 28nd February 2015.

I am pleased to announce Sara Cosgrove, Johns Hopkins University School of Medicine, USA; Jan Kluytmans, Erasmus Medical University, The Netherlands; Sally Roberts, Auckland City Hospital, New Zealand; and Gunnar Kahlmeter, Central Hospital, Sweden will be participating at the meeting. Sara will be presenting the plenary "Multiple Prongs of Stewardship: Less is More – Debunking Stewardship Myths", while Jan and Sally will be presenting "Resistance Links to Animals" and "Improving Care: Infection Prevention and Patient Safety" respectively. In addition to presenting the plenary "EUCAST and Beyond", Gunnar with Erika Matusckek will be presenting two EUCAST workshops on susceptibility testing.

The 2015 Howard Florey Oration will be delivered by Benjamin Howden from Melbourne University, Victoria. Ben will be presenting the talk "Vancomycin and *Staphylococcus aureus* – A Complex Relationship"

The programme's symposia cover many different aspects on antimicrobials and sessions include "Alternative Perspectives on Antimicrobial Use", "Carbapenemases", "What to Report and How to Treat", "Enterococci" and "SMART Platforms". In addition we have two pharmacy symposia on Saturday afternoon titled "The Bugs and Treatment" and "Antimicrobial Stewardship". Six proffered papers and two poster sessions are also planned for the meeting.

To promote discussion and interaction between delegates and the invited speakers the meeting's registration includes lunches, morning and afternoon teas and admission to the Howard Florey Reception and the Industry Reception. I am confident that you will find the meeting's programme both scientifically stimulating and informative and we look forward to meeting you in Brisbane.

Important Dates
Abstract Submission Deadline: Friday 12th December 2014
Early Bird Registration Deadline: Friday 2nd January 2015

Thomas Gottlieb
President ASA

EXCERPTS FROM THE 16TH INTERNATIONAL SYMPOSIUM ON STAPHYLOCOCCI AND STAPHYLOCOCCAL INFECTIONS (ISSSI), CHICAGO, 2014

Joshua Ramsay, School of Biomedical Sciences, Curtin Univeristy.
Matthew O'Sullivan, Centre for Infectious Diseases and Microbiology Laboratory Services, Westmead Hospital.

This occasion, **Joshua Ramsay**, School of Biomedical Sciences, Curtin University, and **Matthew O'Sullivan**, Centre for Infectious Diseases and Microbiology Laboratory Services, Westmead Hospital, provide a summary and their own perspectives of some of the highlights from the 2014 ISSSI conference.

Joshua Ramsay writes:
The 2014 International Symposium on Staphylococci and Staphylococcal Infections (ISSSI) was held in Chicago at the famous Palmer House Hotel in downtown Chicago, in association with the University of Chicago. The Symposium encompassed a broad selection of topics on the molecular biology, antibiotic resistance, genetics and epidemiology of staphylococci infections in humans and other animals. Some of the more intriguing topics discussed were:

The tug-of-war between *S. aureus* and the immune system
Staphylococcus aureus produces a plethora of immunomodulatory proteins to progress infection. Neutrophils secrete a variety of serine protease enzymes (NSPs) such as neutrophil elastase, proteinase 3 and cathepsin G. These NSPs are not directly toxic towards *S. aureus*, however they do degrade virulence factors secreted by *S. aureus* and are thus critical for clearance of infections. Dr Rooijakkers presented research demonstrating that the *S. aureus* extracellular adherence protein Eap and paralogues EapH1 and EapH2, are specific NSP inhibitors. Dr Rooijakkers hypothesises that *S. aureus* secretes these protease inhibitors to prevent neutrophils from degrading *S. aureus* virulence factors during infection. Additionally, as NSPs are important mediators of the inflammatory response, these *S. aureus* Eaps highlight possible drug targets for inflammation control.

Several advanced microscopy techniques were showcased at ISSSI but none were quite as striking as the intravital imaging of *S. aureus* growing within Kupffer cells (KCs) in live mouse liver, presented by Bas Surewaard. KCs are liver macrophages that play an important role in pathogen clearance. The intravital technique videoed labelled *S. aureus* growing and dividing within KCs *in vivo*. Most *S. sureus* were rapidly taken up and killed by KCs, however in ~10% of KCs, *S. aureus* replicated rapidly, undetcted by neutrophils darting around nearby, until KCs were eventually lysed. Living inside KCs not only protected *S. aureus* from neutrophils but also vancomycin.

Wall teichoic acids
Wall teichoic acids (WTAs) are charged glycopolymers found in the exposed cell wall of *S. aureus*. However unlike lipoteichoic acids, they are covalently attached to peptidoglycan rather than embedded in the phospholipid membrane. WTAs, being directly exposed on the surface of *S. aureus*, interact with complement, antibodies, MHC II and CD1. Dr Andreas Peschel reviewed recent research that demonstrated WTAs are critical for adhesion to receptors found on nasal epithelial cells and nasal colonisation. WTAs differed between staphylococcal lineages; Major clonal *S. aureus* lineages produced poly-ribitol-phosphate WTAs, while *S. aureus* ST395 and coagulase-negative staphylococci produced poly-glycerol-phosphate WTA. Susceptibility to infection by bacteriophage was dependent the WTA type, suggesting these molecules restrict horizontal gene transfer between strains with different WTAs.

Antimicrobial synergies and an inverse relationship with virulence.
A reoccurring theme was the synergy between antimicrobial agents with distinct targets. Dr Robert Hancock presented demonstrated effective clearance of *S. aureus* biofilms with the synthetic cationic peptide DJK5 and furthermore that DJK5 significantly lowered the concentration of vancomycin required to treat MRSA biofilms. Prof. Michael Rybak and presented results from a large MIC analysis of strains and demonstrated that high daptomycin and vancomycin resistance is inversely associated with β-lactam resistance and that combination therapy with vancomycin and ceftaroline looks promising.

George Sakoulas also presented data showing that increased vancomycin resistance of *Staphylococcus* is associated with decreased septic shock and inflammatory response. Furthermore he suggested that staphylococci of endocarditis patients may already be primed for daptomycin resistance due to their exposure to natural cationic peptides produced by the human immune system.

Ruth Massey revealed an inverse relationship between methicillin resistance and virulence. MRSA were less virulent than MSSA isolates and this relationship was associated with carriage of the *mecA* gene. Consistent with these data, oxicillan induced *mecA* expression and reduced expression of phenol-soluble modulins (PSMs). Her group found that *mecA* expression interfered with the *agr* regulatory system by reducing cell sensitivity to the quorum-sensing signalling peptide AIP.

EXCERPTS FROM THE 16TH INTERNATIONAL SYMPOSIUM ON STAPHYLOCOCCI AND STAPHYLOCOCCAL INFECTIONS (ISSSI), CHICAGO, 2014 (CONT'D)

Joshua Ramsay, School of Biomedical Sciences, Curtin Univeristy.
Matthew O'Sullivan, Centre for Infectious Diseases and Microbiology Laboratory Services, Westmead Hospital.

Staphylococcal biofilms

Dr Blaise Boles group found that when *Staphylococcus* is grown in the presence of peptone, NaCl and glucose, it produces biofilms resistant to SDS, protease and DNAse treatment. The biofilm matrix was found to be supported by amyloid-like fibres made of PSMs. In this form, the PSMs were unable to cause hemolysis.

Dr Jakub Kwiecinski revealed that fibrin was a major component of *in vivo* biofilms and that the secreted *S. aureus* staphylokinase inhibited biofilm formation. Plasminogen activation by staphylokinase led to cleavage of fibrin and dispersal of biofilms. It is therefore possible that activation of fibrinolysis through pharmacological means may offer potential treatment for *in vivo* biofilms.

Genomic portraits and epidemiology of *Staphylococcus aureus* in humans and livestock

Several large genome sequencing and genome-wide association studies (GWAS) were presented, providing some insight into the diversity and evolution of *Staphylococcus* species. Dr Anne-Catrin Uhlemann presented her research on the evolution of the widespread MSSA clone ST398. Their evidence suggested that ST398 diverged ~40 years ago and that MRSA ST398 evolved from MSSA. Isolates exhibited high SNP diversity and interestingly, lower numbers of mobile genetic elements were observed in non-livestock clones.

Dr Matthew Holden presented an analysis of the evolution of the oldest pandemic MRSA clone ST239/CC8. Consistent with Dr Uhlemann's data, phylogenetic distances between major sequence types/clonal complexes were large and SNP variation within individual clonal complexes was high. Plasmids carrying resistance to hospital disinfectants, and the staphylococcal cassette chromosome-encoded arginine catabolic mobile element (ACME), were associated with ST239. An analysis of isolate transmission between patients suggested that persistently infected patients repeatedly transmitted their strain in a hospital setting. A mother-newborn transmission study presented by Dr Regev-Yochay mirrored these results, revealing frequent and repeated transmission from mother to newborn. Unexplainably there was a significant reduction in SNP variation in *S. aureus* clones isolated from newborns compared to the related clones in the mother.

Dr Ross Fitzgerald presented data from the sequencing of over 800 isolates from 44 different hosts. Phylogenetic analysis suggests that humans are the original host and that multiple host jumps for multiple sequence types/clonal complexes have occurred throughout history. Some clonal complexes appeared to jump hosts more readily, but there was no clear rule-of-thumb. Analysis of MGEs across these isolates revealed an overrepresentation of particular mobile genetic elements with specific hosts. As an example he revealed that MGE-borne cadmium-resistance genes are overrepresented in pigs.

MATTHEW O'SULLIVAN writes:

Once again, the ISSSI brought together researchers from all over the world to discuss progress and to highlight aspects of staphylococcal pathogenesis, evolution, disease and treatment. A number of key "take home messages" are presented here and key references provided.

1. Tim Foster from Trinity College Dublin discussed the role of surface proteins in *S. aureus* pathogenesis. Previously simplified as microbial surface components recognising adhesive matrix molecules (MSCRAMMs), in recent years, the function of *S. aureus* surface proteins have been further elucidated, and it is now known that they have roles in iron scavenging (iron-regulated surface determinant [IsD] proteins), intracellular invasion (fibronectin binding protein) and survival (adenosine synthase [AdsA]), biofilm formation, inflammation, as well as the previously recognized roles of adherence and immune evasion.[1]

2. Suzan Rooijakkers from University Medical Centre Utrecht, the Netherlands spoke about the effect of neutrophil proteases on host defence against *S. aureus* infection, and the role of *S. aureus* extracellular adherence proteins (EAP, and its homologues EAPH1 and EAPH2), which occlude the catalytic site of neutrophil proteases. [2]. Joshua Ramsay has referred to this disucssion above.

3. Sandip Datta from the Bacterial Pathogenesis Unit at the NIH spoke about the role of host cytokines in *S. aureus* skin infection. It appears that the homeostatic cytokines IL-19, IL-20 and IL-24 play a dysregulatory role in this condition. Knockout mice studies give evidence that interrupting the signaling pathway of these cytokines reduces the severity of such infections. [3]

EXCERPTS FROM THE 16TH INTERNATIONAL SYMPOSIUM ON STAPHYLOCOCCI AND STAPHYLOCOCCAL INFECTIONS (ISSSI), CHICAGO, 2014 (CONT'D)

Joshua Ramsay, School of Biomedical Sciences, Curtin Univeristy.
Matthew O'Sullivan, Centre for Infectious Diseases and Microbiology Laboratory Services, Westmead Hospital.

4. Intravital imaging as indicated above also by Joshua Ramsay, (multichannel fluorescent spinning disc confocal microscopy) allows the direct visualization of live footage of tissues at a microscopic level. Using fluorescent dyes, the interaction of individual organisms of *S. aureus* with Kupffer cells in the liver can be directly visualized in a blood stream infection model. This clearly demonstrated that not only are *S. aureus* organisms taken up by Kupffer cells in significant numbers, the organism is also able to continue to replicate in the phagolysosome of these cells. Furthermore, the observation that intracellular *S. aureus* organisms are protected from vancomycin, perhaps given an indication as to why prolonged intravenous antibiotics are required for cure in *S. aureus* bloodstream infection. Interestingly, the technique was also used to demonstrate that 'vancozomes' (vancomycin loaded liposomes) were able to deliver vancomycin into the kupffer cells, resulting in better bacterial killing, also reflected by lower ALT enzyme levels in mice treated with vancozomes compared with conventional vancomycin.

5. The presentation by Andreas Peschel from the University of Tübingen, Germany on *S. aureus* cell wall teichoic acids gave insights of the role of these molecules in epithelial binding and nasal colonisation (through scavenger scavenge-like molecules). These molecules are also targets for host immune cell interactions. Recently, they have been found to be important for horizontal gene transfer, with bacteriophage specificity related to cell wall techoic acid structure. A divergent clade of *S. aureus* (ST395) has been shown to have a unique techoic acid structure, and as such does not undergo horizontal gene transfer with other *S. aureus* lineages, but can exchange DNA with some coagulase negative staphylococci and *Listeria* sp. [4, 5]

6. Robert Hancock, Unversity of British Columbia, discussed promising work on the activity of cationic peptides on MRSA biofilms. Synthetic peptides based on these host defence molecules have been shown to have synergies with conventional antibiotics, in reducing both the MIC of the organism and the amount of *in vitro* biofilm production. The spectrum of activity of these molecules is broad, affecting both Gram negative and Gram positive bacteria. [6]

7. A session on livestock associated *S. aureus* infections explored both the emergence of *mec*C MRSA clones and the relationship between CC398 human and animal clones. Mark Holmes from the department of Veterinary Medicine, University of Cambridge, described the findings of genomic studies of *mec*C MRSA. Some clades of *mec*C MRSA seem to associate geographically rather than according to host, and appear to be able to infect multiples hosts without any significant alteration in their genome. [7] Marc Stegger from the Statens Serum Institute, Denmark presented data from whole genome sequence analysis of the small number of CC398 *S. aureus* isolated from animals and humans in Australia and New Zealand. This provided evidence that these isolates were derived from multiple introductions, rather than any sustained local transmission. [8-10]

8. A session on antimicrobial resistance explored the relationship between resistance to vancomycin and resistance to daptomycin, and the therapeutic options to treat resistant infections. Michael Rybak from the Wayne State University, Detroit, discussed some of the postulated molecular mechanisms of resistance and how resistance to the two agents may be related. Dosing strategies of daptomycin were discussed, with *in vitro* evidence that higher doses of daptomycin in salvage treatment after vancomycin failure may be worth exploring, and that higher dose daptomycin followed by subsequent de-escalation may result in better bacterial killing than the reverse strategy. [11, 12] Resistance to these agents is frequently associated with an increase in susceptibility to beta-lactams (the so called "see-saw" effect), which underlies the rationale for the use of beta-lactams in combination with vancomycin or daptomycin for treating serious or refractory MRSA infections. While ceftaroline (with its inherent activity against MRSA) is an obvious choice for use in such combinations [13], there is also evidence for activity of other beta-lactams (such as oxacillin, nafcillin, cephazolin) in this setting. It seems that exposure to beta-lactam antibiotics leads to changes in the bacterial cell wall charge and thickness that promote daptomycin and vancomycin binding. [14] George Sakoulas from University of California San Diego School of Medicine discussed how daptomycin resistance may be a function of prolonged infection and exposure to host defense peptides, rather than simply drug exposure, and further discussed the rationale for combination therapy. [15]

Antibiotic resistance was further explored in a session on the seemingly inverse relationship between resistance and virulence. Anton Peleg from Monash University described work on elucidating mutations found in isogenic pairs of *S. aureus* isolates from patients with persisting bacteraemic infections exhibiting *in vivo* resistance development. In particular, loss of function mutations in the serine/threonine phosphatase gene *stp1* lead to changes in cell wall metabolism (contributing to daptomycin/vancomycin resistance) but concurrently causing a downregulation of virulence determinant expression such as phenol-soluble modulin, delta haemolysin and superantigen-like proteins [16].

EXCERPTS FROM THE 16TH INTERNATIONAL SYMPOSIUM ON STAPHYLOCOCCI AND STAPHYLOCOCCAL INFECTIONS (ISSSI), CHICAGO, 2014 (CONT'D)

Joshua Ramsay, School of Biomedical Sciences, Curtin Univeristy.
Matthew O'Sullivan, Centre for Infectious Diseases and Microbiology Laboratory Services, Westmead Hospital.

Finally, Ruth Massey, University of Bath specifically focused on the relationship between the activity of cytolytic exotoxins of *S. aureus* isolates and methicillin resistance. In general there seems to be an inverse relationship between cytolytic activity and oxacillin MIC, which seems to be linked to PBP2a production, since knocking out *mecA* in isolation leads to an increase in cytolytic activity. However the relationship between *mecA* expression/oxacillin MIC does not appear to be the same for all cytolytic toxins – exposure to oxacillin and upregulation of *mecA* expression seems to decrease phenol soluble modulin production, but increases panton-valentine leukocidin and alpha toxin production. [17]

Further Reading

1. Foster, T.J., *et al*., *Adhesion, invasion and evasion: the many functions of the surface proteins of Staphylococcus aureus.* Nat Rev Micro, 2014. **12**(1): p. 49-62.
2. Stapels, D.A.C., et al., *Staphylococcus aureus secretes a unique class of neutrophil serine protease inhibitors.* Proceedings of the National Academy of Sciences, 2014. **111**(36): p. 13187-13192.
3. Myles, I.A., et al., *Signaling via the IL-20 receptor inhibits cutaneous production of IL-1[beta] and IL-17A to promote infection with methicillin-resistant Staphylococcus aureus.* Nat Immunol, 2013. **14**(8): p. 804-811.
4. Weidenmaier, C. and A. Peschel, *Teichoic acids and related cell-wall glycopolymers in Gram-positive physiology and host interactions.* Nat Rev Micro, 2008. **6**(4): p. 276-287.
5. Winstel, V., et al., *Wall teichoic acid structure governs horizontal gene transfer between major bacterial pathogens.* Nat Commun, 2013. **4**.
6. Reffuveille, F., et al., *A Broad-Spectrum Antibiofilm Peptide Enhances Antibiotic Action against Bacterial Biofilms.* Antimicrobial Agents and Chemotherapy, 2014. **58**(9): p. 5363-5371.
7. Paterson, G.K., E.M. Harrison, and M.A. Holmes, *The emergence of mecC methicillin-resistant Staphylococcus aureus.* Trends in Microbiology, 2014. **22**(1): p. 42-47.
8. Groves, M.D., et al., *Staphylococcus aureus ST398 detected in pigs in Australia.* Journal of Antimicrobial Chemotherapy, 2014. **69**(5): p. 1426-1428.
9. Jordan, D., et al., *Carriage of methicillin-resistant Staphylococcus aureus by veterinarians in Australia.* Australian Veterinary Journal, 2011. **89**(5): p. 152-159.
10. Williamson, D.A., et al., *Emergence and molecular characterization of clonal complex 398 (CC398) methicillin-resistant Staphylococcus aureus (MRSA) in New Zealand.* Journal of Antimicrobial Chemotherapy, 2014. **69**(5): p. 1428-1430.
11. Rose, W.E., et al., *Daptomycin Activity against Staphylococcus aureus following Vancomycin Exposure in an In Vitro Pharmacodynamic Model with Simulated Endocardial Vegetations.* Antimicrobial Agents and Chemotherapy, 2008. **52**(3): p. 831-836.
12. Vidaillac, C., M.E. Steed, and M.J. Rybak, *Impact of Dose De-Escalation and Escalation on Daptomycin's Pharmacodynamics against Clinical Methicillin-Resistant Staphylococcus aureus Isolates in an In Vitro Model.* Antimicrobial Agents and Chemotherapy, 2011. **55**(5): p. 2160-2165.
13. Barber, K.E., et al., *Observation of "seesaw effect" with vancomycin, teicoplanin, daptomycin and ceftaroline in 150 unique MRSA strains.* Infect Dis Ther, 2014. **3**(1): p. 35-43.
14. Dhand, A., et al., *Use of Antistaphylococcal β-Lactams to Increase Daptomycin Activity in Eradicating Persistent Bacteremia Due to Methicillin-Resistant Staphylococcus aureus: Role of Enhanced Daptomycin Binding.* Clinical Infectious Diseases, 2011. **53**(2): p. 158-163.

EXCERPTS FROM THE 16TH INTERNATIONAL SYMPOSIUM ON STAPHYLOCOCCI AND STAPHYLOCOCCAL INFECTIONS (ISSSI), CHICAGO, 2014 (CONT'D)

Joshua Ramsay, School of Biomedical Sciences, Curtin Univeristy.
Matthew O'Sullivan, Centre for Infectious Diseases and Microbiology Laboratory Services, Westmead Hospital.

15. Mishra, N.N., et al., *Emergence of Daptomycin Resistance in Daptomycin-Naïve Rabbits with Methicillin-Resistant Staphylococcus aureus Prosthetic Joint Infection Is Associated with Resistance to Host Defense Cationic Peptides and mprF Polymorphisms.* PLoS ONE, 2013. **8**(8): p. e71151.

16. Cameron, D.R., et al., *Serine/Threonine Phosphatase Stp1 Contributes to Reduced Susceptibility to Vancomycin and Virulence in Staphylococcus aureus.* Journal of Infectious Diseases, 2012. **205**(11): p. 1677-1687.

17. Rudkin, J.K., et al., *Oxacillin Alters the Toxin Expression Profile of Community-Associated Methicillin-Resistant Staphylococcus aureus.* Antimicrobial Agents and Chemotherapy, 2014. **58**(2): p. 1100-1107.

2014 - 2015 MEETING CALENDAR

2014

International Meeting on Emerging Diseases and Surveillance
31 Oct – Nov 3, Vienna, Austria
website: http://imed.isid.org

15th Asia Pacific congress of Clinical Microbiology and Infection.
Nov 26-29, Kuala Lumpur
Website: http:/www.apccmi2014.org/

2015

Microbiome Forum Asia
19-20 January, Kuala Lumpur, Malaysia
Website: www.globalengage.co.uk

Antimicrobials 2015
February 26-28, Brisbane, Queensland
Website: www.asainc.net.au

Pathology Update 2015
Feb 27 – Mar 1, Melbourne, Victoria
Website: www.rcpa.edu.au

ASID Annual Meeting
Mar 18-21, Auckland, New Zealand
Website: www.asid.net.au

The 2015 TB Summit
March 24-26, London, UK
Website: https://www.regonline.co.uk/

7th international Congress of the Asia Pacific Society for Infection Control
26-29 March 2015, Taipei, Taiwan
website: www.apsic2015.org

25th European Congress of Clinical Microbiology and Infectious Diseases (ECCMID 2015)
25 - 28 April 2015, Copenhagen, Denmark
Website: http://escmid.org/dates_events/

19th Congress of the International Society for Human and Animal Mycology
4-8 May 2015, Melbourne, Australia
Website: www.isham2015.com.au

115th Annual General Meeting, American Society for Microbiology
May 30 – June 5, New Orleans, USA
Website: http://www.asm.org

International Conference in Prevention and Infection Control
16-19 June, Geneva, Switzerland
Website: http://www.icpic2015.com

Australian Society for Microbiology Annual Meeting
12 -15 July, Canberra, ACT
Website: www.theasm.org.au

55th ICAAC
18-21 Sept, San Diego, USA
Website: http://www.asm.org

In 2015 ICCAC will be a joint meeting with the International Society for Chemotherapy

2016

Antimicrobials 2016
February 25-27, Melbourne, Victoria
Website: www.asainc.net.au

26th European Congress of Clinical Microbiology and Infectious Diseases (ECCMID 2015)
9-12 April 2016, Instanbul, Turkey
Website: http://escmid.org/dates_events/

16th Asia Pacific Conference on Clinical Microbiology and Infection (APCCMI)
30 Nov- 3 Dec, Melbourne, Australia
Website: http://www.asainc.net.au

In 2016, the ASM general meeting and ICAAC will be co-located in Boston, June 2016. No organisational details available as on Oct 3 2014.